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ORIGINAL

Characterization of acute renal failure due to cisplatin in cancer patients

Caracterización de la insuficiencia renal aguda por cisplatino en pacientes oncológicos

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ABSTRACT

Introduction: cisplatin is the most nephrotoxic antineoplastic drug and the main cause of acute renal failure in cancer patients.
Objective: to characterize acute renal failure due to cisplatin in oncology patients treated in the Nephrology clinic belonging to the "III Congreso" Hospital in the period 2020-2022.
Method: an observational, descriptive and cross-sectional study was carried out in oncological patients with acute renal failure due to cisplatin, belonging to the Nephrology clinic of Hospital III Congreso, Pinar del Río city, during the period 2020-2022. Variables were defined as: sex, age, presence of active oncological disease, presence of acute renal failure, stage of acute renal failure, urinary volume, urinary sediment, risk factors. The universe was made up of 52 patients, taking as a sample 23 who met the inclusion criteria. The collection of information took into account theoretical, empirical and statistical methods.
Results: male patients aged ≥ 60 years predominated. There was a higher frequency of active neoplastic disease in relation to ARF. Patients who presented ARF predominated for 52,17 % and in Stage II of the disease, with a urinary volume of 0,5-3 L/24 hours. Active urinary sediment predominated for 52,17 %. Hypovolemia was the risk factor most associated with ARF, for 39,13 %.
Conclusions: acute kidney damage, both functional and structural, should be studied more precisely in a cancer patient subjected to toxic drug attacks, in order to prevent kidney consequences.

Keywords: Acute Kidney Failure; Cisplatin; Cancer.

RESUMEN

Introducción: el cisplatino es el fármaco antineoplásico más nefrotóxico y la principal causa de insuficiencia renal aguda en pacientes oncológicos.
Objetivo: caracterizar la insuficiencia renal aguda por cisplatino en pacientes oncológicos atendidos en la consulta de Nefrología perteneciente al Hospital "III Congreso" en el período 2020-2022.
Método: se realizó un estudio observacional, descriptivo y transversal en pacientes oncológicos con insuficiencia renal aguda por cisplatino, pertenecientes a la consulta de Nefrología del Hospital III Congreso, ciudad Pinar del Río, durante el período 2020-2022. Se definieron variables como: sexo, edad, presencia de enfermedad oncológica activa, presencia de insuficiencia renal aguda, estadio de insuficiencia renal aguda, volumen urinario, sedimento urinario, factores de riesgo. El universo estuvo integrado por 52 pacientes, tomándose como muestra 23 que cumplieron con los criterios de inclusión. La recolección de la información tuvo en cuentas métodos teóricos, empíricos y estadísticos.
Resultados: predominaron los pacientes del sexo masculino y edad ≥ 60 años. Existió mayor frecuencia de enfermedad neoplásica activa en relación con IRA. Los pacientes que presentaron IRA predominaron para

un 52,17 % y en el Estadio II de la enfermedad, con un volumen urinario de 0,5-3 L/24 horas. Predominó el sedimento urinario activo para un 52,17 %. La hipovolemia fue el factor de riesgo que más se asoció a IRA, para un 39,13 %.
Conclusiones: se debe estudiar con más precisión, el daño renal agudo, tanto funcional como estructural, en un paciente con cáncer sometido a agresiones tóxicas medicamentosas, con la finalidad de prevenir las consecuencias renales.

Palabras clave: Insuficiencia Renal Aguda; Cisplatino; Cáncer.

INTRODUCTION

Acute kidney injury is a potentially fatal pathological process that occurs in approximately 5 % of all hospitalized patients and up to 30 % of intensive care unit admissions.[1] Patients with AKI, regardless of their associated comorbid conditions, have a mortality rate more than five times higher. The hallmark of AKI is a sudden reduction in the glomerular filtration rate (GFR), resulting in the retention of nitrogenous waste products (creatinine, blood urea nitrogen [BUN], and other molecules that are not routinely measured). In the early stages of AKI, patients are often asymptomatic, and the condition is only diagnosed by observed elevations in BUN and serum creatinine levels or oliguria.[2] In cancer patients, AKI has a mixed etiology, in which sepsis, ischemia, and nephrotoxicity often coexist and complicate recognition and treatment.[3] The classification of ARF includes prerenal, acute postrenal obstructive nephropathy, and intrinsic acute kidney diseases. Of these, only "intrinsic" ARF represents a true kidney disease, while prerenal and postrenal ARF are the consequence of extrarenal diseases that lead to a decrease in the GFR. Intrinsic ARF involves four structures of the kidney, including tubules, glomeruli, interstitium, and intrarenal blood vessels.[4]

Cancer and platinum complexes

Cancer is the second leading cause of death and morbidity in Europe (3,7 million new cases each year). The average age of patients at the time of cancer diagnosis is 65 years. Of the 47 % of cancer survivors, almost half are 70 years of age or older. Data from other countries show 22 % in the United States, 25 % in Japan, and 15,5 % in Austria. An aging population will increase the number of patients whose cancer will be complicated by other acute (IRA) or chronic (CKD) diseases.[5,6,7]

According to the 2020 Cuban Health Statistics Yearbook, mortality from malignant tumors is the second leading cause of death in the country, with a rate of 223,0 per 100 000 inhabitants, and at the end of 2020, 21,3 % of the national population was in the elderly age group. For this reason, an aging population will increase the number of patients whose cancer will be complicated by other acute or chronic kidney diseases.[8]

The history of platinum as a component of metal complexes with anticancer activity began with the observation of the antineoplastic properties of the cis-diaminodichloroplatinum (II) complex (cisplatin) in 1965. This accidental discovery arose from the study of the effect of electric fields on the growth of *Escherichia coli* (E. coli) bacteria using platinum electrodes. The bacteria stopped dividing one hour after an electric current was passed through the culture. Because chloride and ammonia ions were present in the solution during electrolysis, several platinum complexes were formed, including cis-diaminodichloroplatinum (II), which proved to be the most effective in inhibiting bacterial growth.[6]

Cancer therapies changed radically after the discovery of this complex. In the 1970s, cisplatin was included in numerous treatment protocols as a monotherapy or as part of combination chemotherapy to treat various tumors. The high activity of this drug against urogenital tumors, cervical carcinomas, and other types of tumors such as melanoma, osteosarcoma, and neuroblastoma, as well as the associated toxicity problems (, nephrotoxicity, ototoxicity, emesis, neurotoxicity)[2] prompted the study and development of new-generation platinum-based molecules that were analogous in their antitumor activity but reduced in toxicity. It was then, during the 1970s and 1980s, that new drugs such as carboplatin and oxaliplatin were developed.[3] In March 1989, Bristol-Myers Squibb obtained FDA (Food and Drug Administration) approval to market carboplatin under the trade name Paraplatin®.[7]

Cisplatin and carboplatin are two drugs widely used in the treatment of solid tumors, particularly in ovarian, head and neck, cervical, and lung cancer. Their antineoplastic mechanisms of action are very similar. When activated intracellularly, two valences of the platinum ion are released, forming two stable bonds with the nitrogenous bases of DNA. The result is the alteration of the three-dimensional configuration of the genetic material, the generation of transcription errors, and the inability of the chains to separate for replication. Despite their high effectiveness in the clinical setting, their ability to cause nephrotoxicity and ototoxicity limits their use.[8]

Cisplatin is the most nephrotoxic antineoplastic drug. Approximately 25-30 % of patients treated with it

have been shown to develop acute kidney injury even after a single dose. It can cause mild, transient, and self-repairing episodes of kidney damage that may not have significant health consequences. However, the occurrence of cumulative episodes can lead to more harmful consequences. In fact, repeated cycles of chemotherapy with cisplatin can lead to chronic tubulointerstitial fibrosis. A small fraction of these patients would require lifelong dialysis and would also be at high risk of mortality in the medium and long term. Therefore, early detection of mild forms of AKI is an unmet goal for better management of this disease.[9]

Cisplatin nephrotoxicity: Histopathological aspects

Early clinical experience with cisplatin showed functional toxicity consisting of acute reduction in glomerular filtration, evidenced by decreased creatinine clearance; in the most severe cases, signs of tubular dysfunction were also present. Although the acute profile of the condition and the decrease in glomerular filtration suggested the existence of vascular and glomerular lesions, microscopic studies showed the presence of focal tubular necrosis, predominantly affecting the distal tubules. According to experimental data in animals, however, the lesions were more prominent in the outermost cortex, and more specifically in the proximal tubules. Other authors have observed that the highest degree of necrosis occurs at the cortico-medullary junction, affecting both the proximal tubules and the distal segments of the nephrons, with morphological integrity of the glomeruli.[9]

Electron microscopy shows severe necrotic changes in all tubular segments, mainly in the proximal tubules in rats, or more pronounced in the distal and collecting tubules in humans. Noteworthy features include an increase in the number of lysosomes, structural abnormalities of the mitochondria, and loss of the brush border, but there are also nuclear atypia, changes in glycogen, and dense deposits in the Bowman's capsule that must correspond to platinum accumulations.[10]

Another factor to consider is the different susceptibility of each experimental animal to the drug. In dogs and occasionally in humans, tubular damage has been reported to reach a high degree of severity even before glomerular filtration is impaired. This would suggest that renal failure is secondary to obstruction of the tubular lumen by necrotic debris or to a vasoconstrictor mechanism mediated by the juxtaglomerular apparatus via the renin/angiotensin axis, which would be triggered by excess sodium not reabsorbed in the proximal tubule. However, in rats and in most clinical experiences, alterations in hemodynamics and glomerular filtration appear to occur before tubular necrosis occurs, raising doubts about the contribution of tubular damage to the pathogenesis of this type of renal failure.[10,11]

There may even be differing interpretations for simple reasons related to experimental technique. For example, adult rats have been reported to be more susceptible than young rats to cisplatin nephropathy, but in humans the opposite may be true.

With regard to acute renal histological changes, in the 96-hour period immediately following the first treatment cycle, there is a combination of glomerular dysfunction and cessation of tubular functions.[11]

Most authors believe that repeated cycles of cisplatin are accompanied by a gradual reduction in glomerular filtration, and a decrease in the kidney's ability to excrete the free fraction of this drug has been reported, which could be explained by impaired glomerular filtration, but also by damage to the enzymatic mechanisms responsible for tubular secretion of the drug.[9,10,11,12]

Cisplatin nephrotoxicity: Hemodynamic effects

Very early on, there is a decrease in renal plasma flow followed by a decrease in the glomerular filtration rate; and this glomerular failure is an acute phenomenon with permanent, non-progressive sequelae, but its pathogenesis is subject to debate. One of the determining factors appears to be selective vasoconstriction of the afferent arteriole to the glomerulus.

It has been suggested that the underlying mechanism consists of stimulation of the renin/angiotensin axis, followed by an increase in renin and subsequent hyperactivity of angiotensin II, which acts on the glomerular arterioles via a calcium channel-coupled receptor. However, it is not clear whether renin release is due to a direct toxic effect of cisplatin or a sequela of tubulopathy. Incidentally, tubular injury associated with the use of this drug is accompanied by acute polyuria, which plunges the body into a state of dehydration and renal hypoperfusion, perhaps through a feedback mechanism in which prostaglandins may play a role.

The drop in glomerular filtration could also be explained by vascular mechanisms, either due to spastic angiopathy attributable to cisplatin or through the local stimulation of reactions involved in blood coagulation.[13]

Cisplatin nephrotoxicity: Tubular effects

From a histopathological point of view, the morphological preservation of the glomeruli is striking in this toxic nephropathy, alongside florid lesions corresponding to classic acute tubular necrosis (ATN). The topography varies from one species to another and the consequences are also variable. It has been noted that the cells undergo "ballooning" which, due to a simple space conflict, can lead to ischemia and perhaps

reactive glomerular vasoconstriction. Cell debris also induces obstruction of the tubular lumen, secondarily compromising glomerular function. However, it is commonly accepted that the interruption of glomerular filtration is a largely independent phenomenon and that the severity of ATN does not correlate well with the degree of uremia but does correlate with the appearance of various electrolyte disturbances, notably hypomagnesemia.[14,15,16]

Cisplatin enters the renal parenchyma as a free drug; a fraction reaches the tubular cell through its luminal pole, as cisplatin is filtered in the glomerulus and reabsorbed by an organic anion transport system (OCT2); another portion is eliminated by active tubular secretion, using an organic cation transport system (OCT). Once this drug penetrates the tubular cell, or even the tubular lumen, it undergoes rapid molecular transformations, caused by detoxification reactions and by harmful processes conditioned by the pH and chlorine concentration in the medium, which generate hyperreactive chemical species that are believed to be responsible for cytotoxicity. The nephrotoxic molecules interact with subcellular structures distributed throughout the nucleus, cytosol, cell membranes, and various organelles.[17]

On the other hand, mitochondria have been proposed as targets for cisplatin, due to their remarkable ultrastructural changes in cases of nephropathy caused by this drug. This drug has been said to disrupt cellular respiration reactions and induce the release of mitochondrial calcium, which is why it could affect vital cellular processes such as the functioning of ATP-dependent ion pumps, the stability of lysosomes, or the polymerization of cytoplasmic microtubules. Cisplatin has also been reported to alter the function of membrane ion pumps in otic cells and in the brush border of renal tubule cells. Finally, it is argued that it could damage the proximal tubule through lysosomal membrane instability, releasing autolytic enzymes.[18,19]

Whatever the intimate action of cisplatin, it is a fact that sodium absorption in the proximal tubule and chlorine absorption in the loop of Henle are interrupted.

On the one hand, urinary osmolarity increases, leading to polyuria, which only aggravates the impairment of glomerular filtration; and on the other hand, the absorption of calcium and magnesium, which are normally coupled with that of chlorine in the ascending limb of the loop of Henle, is interrupted, without ruling out the loss of cations due to damage to other segments of the nephron.[20]

Scientific problem

Considering the above, the following scientific problem is posed: What are the characteristics of acute renal failure due to cisplatin in cancer patients treated at the Nephrology Clinic of Hospital III Congress in the period 2020-2022?

Research objective:

Cancer patients with acute renal failure due to cisplatin.

Objectives:

General: to characterize acute renal failure due to cisplatin in cancer patients treated at the Nephrology Clinic of Hospital III Congreso between 2020 and 2022.

Specific:

- Identify patients with acute kidney injury and its stage according to diagnostic criteria.
- Determine risk factors for acute kidney injury.
- Establish the relationship between demographic and clinical variables and the biochemical parameters studied.

METHOD

a-Classification of the research

Research – Development.

b- General aspects of the study

Observational, descriptive, and cross-sectional.

An observational, descriptive, and cross-sectional study was conducted in cancer patients with acute renal failure due to cisplatin, belonging to the Nephrology Department of Hospital III Congress, Pinar del Río, during the period 2020-2021.

c-Definition of the study universe and sample

The universe consisted of 53 cancer patients belonging to the Nephrology Department of Hospital III Congress, with a sample of 20 who met the selection criteria.

Selection criteria

Inclusion criteria:

- Patients aged 19 years or older diagnosed with cancer and treated with cisplatin.
- Patients who gave their informed consent to participate in the study.

Exclusion criteria:

- Patients who did not provide informed consent.

Exit criteria:

- Patients who withdrew from the study.
- Patients who became decompensated and/or died during the study.

Techniques for obtaining information:

A data form prepared by the research project team was used for the study patients. The data obtained from this form were collected manually for subsequent tabulation. Information was obtained from the results of the interview, physical examination, and laboratory tests, as well as from the individual medical records and family health histories of the patients.

To meet the specific objectives, the following variables were used:

Table 1. Operationalization of variables

Variable	Classification	Scale	Description	Indicator
1. Sex	Nominal dichotomous qualitative	Male Female	According to biological sex.	Frequency and percentage
2. Age	Continuous quantitative	19 40 ≥60	According to age in years	Frequency and percentage
3. Presence of active cancer	Nominal dichotomous qualitative	Yes No	According to personal medical history	Frequency and percentage
4. Presence of acute renal failure (ARF)	Nominal dichotomous qualitative	Yes No	According to diagnostic criteria	Frequency and percentage
5. Stage of AKI	Qualitative ordinal	I II III	Category based on the AKIN scale	Frequency and percentage
6. Urinary volume	Continuous quantitative	0,5–3 liters >3 liters	According to diuresis in 24 hours	Frequency and percentage
7. Urinary sediment	Qualitative dichotomous nominal	Active sediment Non-active sediment	According to laboratory abnormalities found in urine	Frequency and percentage
8. Risk factors	Nominal polytomous qualitative	No known factors Use of nephrotoxic drugs Sepsis Hypovolemia Renal lithiasis	Depending on whether or not it is present in the patient, or is reported by the patient or their legal representative during the consultation, or is recorded in the medical record.	Frequency and percentage

Statistical data processing

The information collected from the sample was processed using the Systat statistical package, version 9.1. The data obtained were organized and presented in frequency distribution tables based on the objectives set. Summary measures were then calculated for qualitative data (absolute and relative frequencies).

Ethical considerations

This work will be carried out in accordance with medical ethics. Each research subject was given a document, as proof that they agreed to participate in the research after being informed about the purpose of the study,

the procedures that were carried out, the benefits of the research, the alternative of participating or not, the confidentiality of the data obtained, that they were not exposed to additional risks, and the voluntary nature of their participation with oral and written consent. This research was not conducted for profit.

RESULTS

Table 2. Distribution of patients by sex. Hospital III Congreso. 2021-2022

Sex	No	%
f	11	47,83
m	12	52,17
Total	23	100

Of the 23 patients, 12 (52,17 %) were male and 11 (47,83 %) were female.

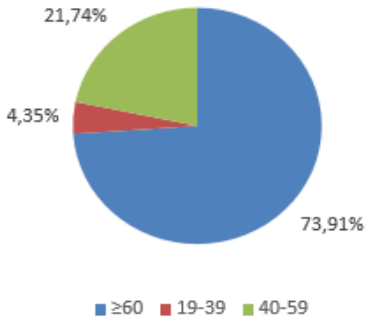

Figure 1. Distribution of patients by age. Hospital III Congreso. 2021-2022

In this study, patients aged ≥ 60 years were more frequent, accounting for 73,91 %.

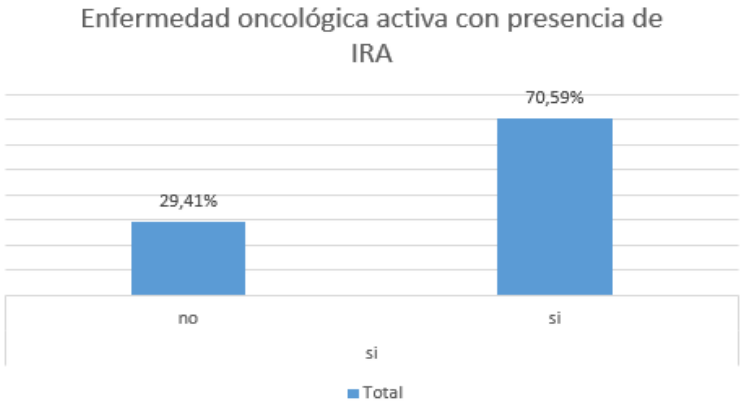

Figure 2. Distribution of patients according to the presence of active cancer related to AKI. Hospital III Congreso. 2021-2022

Active neoplastic disease was predominant in 70,59 % of cases.

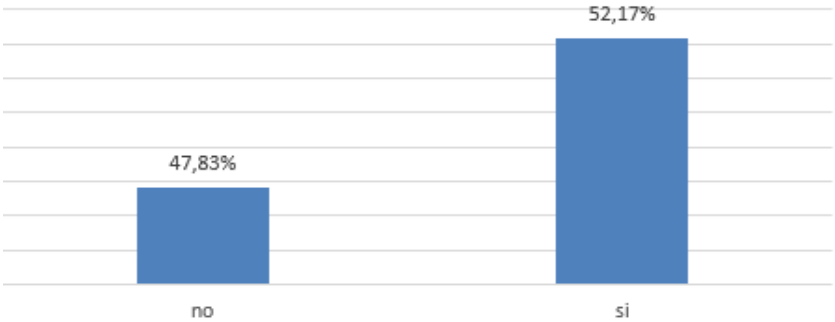

Figure 3. Distribution of patients according to the presence of acute renal failure (ARF). Hospital III Congress. 2021-2022

Patients with ARF predominated at 52,17 %.

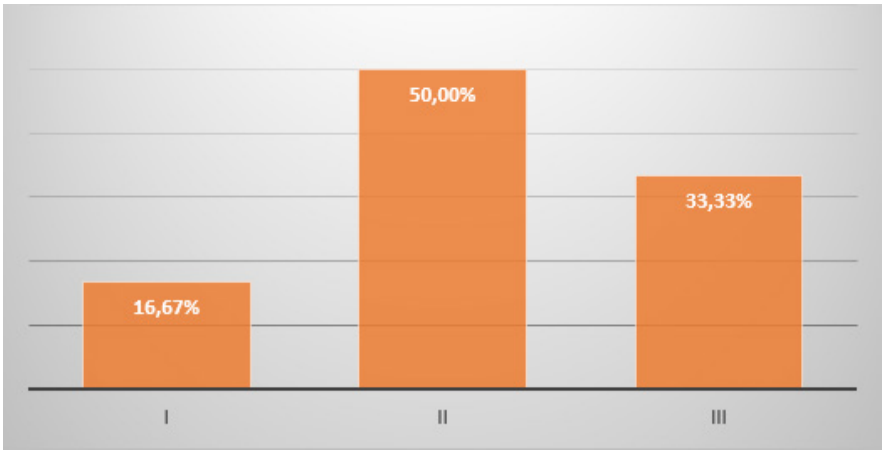

Figure 4. Distribution of patients according to stage of ESRD. Hospital III Congress. 2021-2022

The behavior of acute kidney injury according to its functionality can be observed, where the AKIN classification was used to standardize criteria, with patients in Stage II being the most representative, accounting for 50 % of the total.

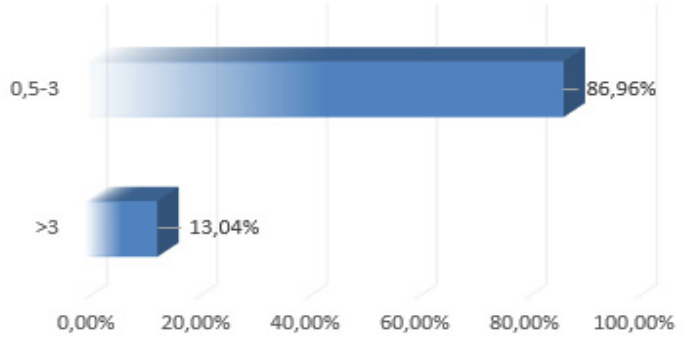

Figure 5. Distribution of patients according to urinary volume. Hospital III Congress. 2021-2022

Patients with a urinary volume of 0,5-3 L in a 24-hour sample predominated, accounting for 86,96 %.

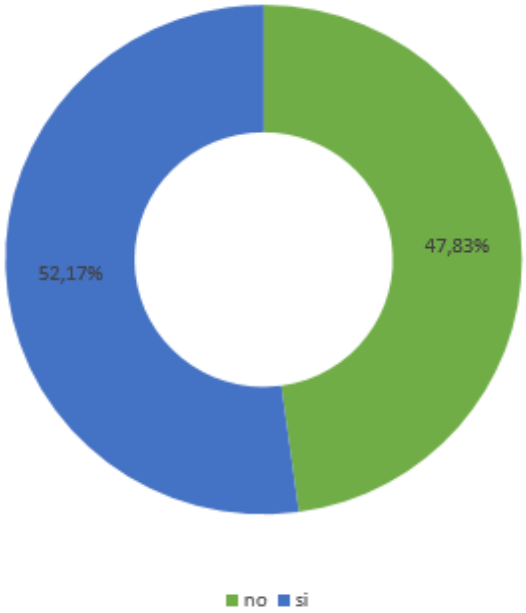

Figure 6. Distribution of patients according to urinary sediment. Hospital III Congreso. 2021-2022

Active urinary sediment predominated in our study, accounting for 52,17 %.

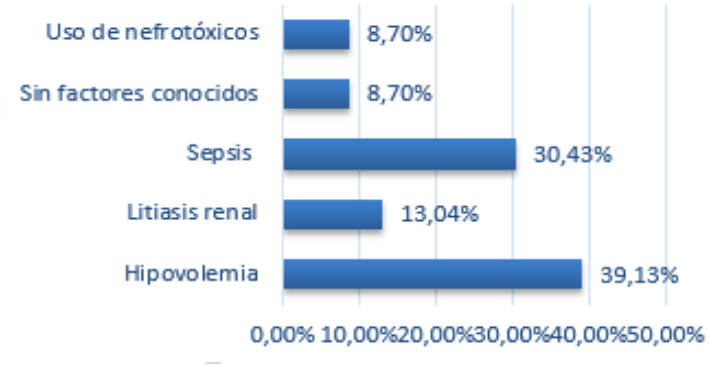

Figure 7. Distribution of patients according to risk factors related to AKI. Hospital III Congress. 2021-2022

In our study, hypovolemia was the risk factor most associated with ARF in patients undergoing antineoplastic treatment with cisplatin, at 39,13 %.

DISCUSSION

Based on the characterization performed, it is valid to note that the data provided by this research is consistent with the study conducted by Heras Benito M et al., which discusses a descriptive cross-sectional study of 163 outpatients with a similar predominance of males (55,2 %).[21,22,23]

Other published studies have not found statistically significant differences in relation to gender.[24,25]

Age is an important factor in the analysis of adverse reactions associated with chemotherapeutic drugs, not only in terms of the incidence of cancer, but also because of physiological changes at certain ages, such as childhood or old age, which generate pharmacokinetic changes associated with the use of these drugs.[26,27]

Most cancers for which alkylating drugs are used as treatment occur in people over 40 years of age. For example, breast cancer occurs more frequently in women over 50, and some cancers, such as prostate cancer, peak in men over 60. Other factors such as comorbidities and polypharmacy may be aggravating factors for the occurrence of adverse reactions, coupled with the fact that with age, humans experience a deterioration in kidney and liver function, which leads to alterations in drug metabolism and a decrease in elimination, prolonging the half-life of drugs, a factor that increases their toxicity.[28]

The study conducted by Heras Benito M et al. agrees with the results of this research, with an average age of 64,58 years.[29]

The results obtained are consistent with several studies.[30,31]

Platinum derivatives are chemotherapy agents widely used in the treatment of solid tumors.[30]

Cisplatin is widely used in the treatment of testicular, ovarian, head, stomach, and lung cancer.[30,31]

Some tumors or antineoplastic agents may also have a deleterious effect on the glomerular structure and cause proteinuria by damaging the podocytes.[29,39,31]

A study by Díaz Mederos et al. showed that 25-30 % of patients develop ARF after the use of alkylating agents (cisplatin).[8]

Despite the potential nephrotoxicity of these drugs, not all patients treated with them end up suffering from ARF; rather, this depends on each individual's susceptibility to the disorder. This predisposition to ARF may be genetic or acquired throughout life and will determine whether patients receiving the same cancer treatment will develop kidney damage.[28,30]

The decrease in glomerular filtration associated with cisplatin toxicity usually occurs 3 to 5 days after exposure. Cisplatin doses greater than 50 mg/m2 are sufficient to cause kidney damage. Kidney damage is typically reversible, but repeated doses of cisplatin greater than 100 mg/m2 can cause irreversible kidney damage.[31,32,33]

These changes and the significant associations observed could be related to the dose used to treat each condition, since it is clear that some neoplasms require higher doses of the drug.[34,35]

The decrease in glomerular filtration associated with cisplatin toxicity usually occurs 3 to 5 days after exposure. Cisplatin doses greater than 50 mg/m^2 are sufficient to cause kidney damage.[36]

The kidney is the main organ responsible for clearing cisplatin. The most common clinical picture is the gradual onset of non-oliguric ARF (urine output > 400 ml/24 hours).

Patients with non-oliguric ARF tend to have a better prognosis, mainly due to less severe injury or a higher incidence of nephrotoxic-induced AKI in the non-oliguric group.[37]

A study by Díaz-Mederos et al. is consistent with the results obtained in our research. Qualitative and quantitative analysis of urine using the Addis count for 8 hours showed active urinary sediment, with a predominance of leukocyturia in 41,2 % of cases.[8]

The increase observed in the urinary excretion of these biomarkers when ARD develops may be due to the fact that platinum agents trigger apoptosis and cell necrosis processes and affect the functioning of the tubular epithelium, causing a defect in its absorption mechanisms.[38]

Similarly, several risk factors for the occurrence of antineoplastic nephrotoxicity have been described, including cumulative and frequent doses of the drug, intrinsic toxicity of the antineoplastic, advanced age, and smoking.[39]

The aim is to identify patients at risk of developing ADR early on, in a more selective and specific manner than with current methods after receiving chemotherapy treatment. To this end, predisposition biomarkers are being investigated, which are defined as molecules associated with defined anatomopathological patterns or pathogenic mechanisms that detect susceptibility to kidney damage and allow patients to be classified on this basis.[39,40]

CONCLUSIONS

Male patients (52,17 %) and patients aged ≥ 60 years (73,91 %) predominated. There was a predominance of active neoplastic disease in relation to ARF in 70,59 % of cases. Patients with ARF predominated at 52,17 %. Patients in Stage II ARF predominated, representing 50 % of the total, with a urine volume of 0,5-3 L in a 24-hour sample for 86,96 %. Active urinary sediment predominated in our study at 52,17 %. Hypovolemia was the risk factor most associated with ARF in patients undergoing antineoplastic treatment with cisplatin, at 39,13 %. Acute renal damage, both functional and structural, should be studied more precisely in cancer patients undergoing toxic drug treatment in order to prevent renal consequences.

BIBLIOGRAPHIC REFERENCES

1. Heras Benito M, Calle García L, Fernández-Reyes L M. Carta al Director A propósito de la onconefrología: enfermedad renal crónica en pacientes oncológicos no hospitalizados. Nefrología. 2020; 40(3):365-6. https://www.revistanefrologia.com/es-a-proposito-onconefrologia-enfermedad-renal-articulo-S0211699519301134.

2. Alonso F, Auñón P, Cavero T, Salgueira M, Praga M, Quiroga B, et al. Consulta monográfica de onconefrología. Justificación y puesta en marcha. Nefrología. 2021; 41(2):154-164. https://www.revistanefrologia.com/en-pdf-S0211699520301715.

3. Finkel KW. Onconefrología:enfermedad renal en pacientes con cáncer. En Schorecki K, Chertow Glenn M, Marsden Philip A, Taal Maarten W, Yu Alan SL, editores. Brenner y Rector. El riñón. Vol 2.10 ed.España:Elsevier;2018.p.1390-1407

4. Izzedine H, Perazella M. Onco-nephrology: An appraisal of the cancer and chronic kidney disease links. Nephrol Dial Transplant. 2015; 30(12):1979-88. file:///C:/Users/laptop/AppData/Local/Temp/ONCKDlink.pdf

5. Li Y, Chen X, Wang Y, Hu J, Xu J, Jiang W, et al. Epidemiology of acute kidney injury and associated factors among patients with malignancy: Analysis of hospital inpatient database in Shanghai, China. J Onco-Nephr. 2019; 3(1). https://journals.sagepub.com/doi/pdf/10.1177/2399369319830096

6. Wong G, Staplin N, Emberson J, Baigent C, Turner R, Chalmers J, et al. Chronic kidney disease and the risk of cancer: An individual patient data meta-analysis of 32,057 participants from six prospective studies. BMC Cancer. 2016. https://www.researchgate.net/publication/305376961_Chronic_kidney_disease_and_the_risk_of_cancer_An_individual_patient_data_metanalysis_of_32057_participants_from_six_prospective_studies/link/5fc2127f458515b7977cb318/download.

7. Ministerio de Salud Pública. Anuario Estadístico de Salud 2019. 48 edición. La Habana; 2020. http://bvscuba.sld.cu/anuario-estadistico-decuba/

8. Díaz-Mederos, E., Peñate-Tamayo, F. D., et al. Consulta monográfica de Onconefrología, experiencia en Villa Clara. Nefrología. https://oncocienfuegos2021.sld.cu/index.php/oncocfg/conferencias/paper/veiew/218/0

9. Conde Sánchez L. Registró Cuba en 2020 un incremento en las personas mayores de 60 años. Granma 2021 Ene 29. Citado 15 julio 2021.

10. Lorenzo V, López Gómez J. Enfermedad Renal Crónica. Nefrología al día. 2021. https://www.nefrologiaaldia.org/es-pdf-enfermedad-renal-cronica-136.

11. Cosmai L, Porta C, Gallieni M, Perazella M. Onco-nephrology: A decalogue. Nephrol Dial Transplant. 2016; 31(4): 519-9. https://air.unimi.it/retrieve/handle/2434/314897/780048/Gallieni_Onco_2016.pdf

12. Capasso A, Benigni A, Capitanio U, Danesh F, Di Marzo V, Gesualdo L, et al. Summary of the International Conference on Onco-Nephrology: An emerging field in medicine. Kidney International. 2019; 96(3):555-67. https://air.unimi.it/retrieve/handle/2434/674370/1382088/Capasso%20_Summary%20of%20the%20International%20Conference%20on%20Onconephrology%20KI.pdf

13. Cosmai L, Porta C, Perazella M, Launay-Vacher V, Rosner M, Jhaveri K, et al. Opening an onconephrology clinic: Recommendations and basic requirements. Nephrol Dial Trasp . 2018]; 33(9):1503-10. https://academic.oup.com/ndt/article/33/9/1503/5049712

14. Arjona-Barrionuevo J, Gonzales Vargas-Machuca M, Guerrero-Márquez F, Gil-Sacaluga L, Gentil-Govantes MA. Utility of a cardiorenal monographic consultation to reduce the cardiovascular mortality of the renal transplant patient. J Cardiol Cardiovasc Sci. 2019; 3(3). https://www.cardiologyresearchjournal.com/articles/utility-of-a-cardiorenal-monographic-consultation-to-reduce-the-cardiovascular-mortality-of-the-renal-transplant-patient.pdf

15. Herrera Valdés R. Enfermedad renal crónica: enfoque sistemico, epidemiológico, clínico e intersectorial. En: Quesada Pantoja J, editor. Nefrología. La Habana: Editorial Ciencias Médicas 2016. p. 102-30.

16. De Francisco A, Macia M, Alonso F, García P, Gutierrez E, Quintana L, et al. Onconefrología: Conexión cáncer y riñón. 2019. Disponible en nefrologia-dia-202.pdf

17. Soukup T, Lamb B, Arora S, Darzi A, Sevdalis N, Green J. Successful strategies in implementing a multidisciplinary team working in the care of patients with cancer: An overview and synthesis of the available literature. J Multidiscip Healthc. 2018; 11:49-61. file:///C:/Users/laptop/AppData/Local/Temp/Successful_strategies_in_implementing_a_multidisci.pdf

18. Sociedad Española de Nefrología.[Internet]. Grupo Español de Onconefrología (ONCONEFRO). 2018. https://www.senefro.org/modules.php?name=grupos&d op=viewgroup&idgroup=14245.

19. Rosner M, Perazella M. Acute kidney injury in patients with cancer. N Engl J Med. 2017. https://www.researchgate.net/publication/326878570_Acute_Kidney_Injury_in_Patients_With_Cancer/link/5d24ce42a6fdcc2462d0517a/download

20. Izzedine H, Perazella M. Anticancer drug-induced acute kidney injury. Kidney Int Rep. 2017. https://www.researchgate.net/publication/313815100_Anticancer_Drug-Induced_Acute_Kidney_Injury

21. Arias M, Moreso F. Proyecto Prometeo 2016: Cáncer en el trasplantado renal: ¿se puede prevenir? Nefrologia [Internet] 2018 [citado 26 Jul 2021]; 9(S1) [aprox. 28 p.]. Available from: https://www.revistanefrologia.com/es-pdf-X2013757518622216

22. Cheung C, Tang S. An update on cancer after kidney transplantation. Nephrol Dial Transplant. 2019; 34. https://academic.oup.com/ndt/article/34/6/914/5107809

23. Pérez-Sánchez L, Gines Casanova A, Morales A. Biomarcadores de nefrotoxicidad capaces de identificar pacientes oncológicos en riesgo de desarrollar daño renal. Farma Journal. 2019; 4(2). https://revistas.usal.es/index.php/2445-1355/article/view/fj2019424150.

24. Perazella MA, Izzadine H. New drug toxicities in the onco-nephrology world. Kidney Int. 2015; 87:909-17.

25. De Francisco A, Arias Guillén M, Pérez-Valderrama B, C S. Post-contrast acute kidney injury in cancer patients. Nefrología. 2019;39. https://www.revistanefrologia.com/en-pdf-S2013251419301610

26. Cosmai L, Porta C, Foramitti M, Perrone V, Mollica L, Gallieni M, et al. Preventive strategies for acute kidney injury in cancer patients. CKJ. 2021; 14(1). doi: 10.1093/ckj/sfaa127

27. Malyszko J, Bamias A, Danesh F, Debska-Slizien A, Gallieni M, Gertz M, et al. Conferencia de Controversias de KDIGO sobre Onconefrología: enfermedad renal en procesos hematológicos malignos y

aumento de cáncer después del trasplante de riñón. Nefrología al día. 2020. https://static.elsevier.es/nefro/monografias/1/369/369_180320212108.pdf.

28. Torres da Costa e Silva V, Costalonga E, Coelho F, Caires R, Burdmann E. Assessment of kidney function in patients with cancer. Adv Chronic Kidney Dis. 2018; 25(1). https://www.ackdjournal.org/action/showPdf?pii=S1548-5595%2817%2930187-8

29. De Francisco A. Efectos renales adversos del tratamiento del cáncer. NefroPlus.2019;11(1):1-16.

30. Sprangers B, Abudayyeh A, Latcha S, Perazella M, Jhaveri K. How to determine kidney function in cancer patients? Eur J Cancer. 2020; 132. https://www.sciencedirect.com/science/article/abs/pii/S0959804920301763

31. Chancharoenthana W, Wattanatorn S, Vadcharavivad S, Eiam-Ong S, Leelahavanichkul A. Agreement and precision analyses of various estimated glomerular filtration rate formulae in cancer patients. Sci Rep. 2019; 9. https://www.researchgate.net/publication/338020226_Agreement_and_Precision_Analyses_of_Various_Estimated_Glomerular_Filtration_Rate_Formulae_in_Cancer_Patients/link/5fb66caa458515b7975127e2/download

32. Ballová V, Provencio Pulla M. ESMO HANDBOOK of cancer treatments in special clinic situations 2013. http://mauriciolema.webhost4life.com/rolmm/downloads/files/ESMOCaRxInSpecClinSit2013.pdf

33. De Francisco A, Macía M, Alonso F, García P, Gutiérrez E, Quintana L, et al. Onco-Nefrología: cáncer, quimioterapia y riñón. Nefrología. 2019; 39(5). https://www.revistanefrologia.com/es-pdf-S021169951930027X.

34. Thavarajah S, Choi M. The Use of Erythropoiesis-Stimulating Agents in Patients With CKD and Cancer: A Clinical Approach. Am J Kidney Dis 2019; 74(5): https://www.ajkd.org/article/S0272-6386(19)30767-X/pdf

35. Escobar Álvarez Y, de las Peñas Bataller R, Perez Altozano J, Ros Martínez S, Sabino Álvarez A, Blasco Cordellat A, et al. SEOM clinical guidelines for anaemia treatment in cancer patients (2020). ClinTransl Oncol. 2021. Available from: https://doi.org/10.1007/s12094-021-02580-2.

36. Z.I. Cherney D, S. Rosenson R, R. Lawler P. Atherosclerotic Cardiovascular Disease and Chronic Kidney Disease. J Am Coll Cardiol. 2019; 73(23). http://www.onlinejacc.org/content/73/23/2971.

37. López Gómez J, Vega Martínez A. Alteraciones Cardiovasculares en la Enfermedad Renal Crónica. Nefrología al día. 2020. https://www.nefrologiaaldia.org/es-pdf-alteraciones-cardiovasculares-enfermedad-renal-cronica-179.

38. Rodrigo Orozco B. Enfermedad cardiovascular (ECV) En la enfermedad renal Crónica (ERC). Rev. méd. Clín.Las Condes. 2015; 26(2). https://www.elsevier.es/es-revista-revista-medica-clinica-las-condes-202-pdf-S0716864015000358.

39. Mendoza-Torres E, Bravo-Sagua R, Villa M, Flores N, José Olivares M, Calle X, et al. Enfermedades cardiovasculares y cáncer: ¿dos entidades mutuamente relacionadas? . Rev Chil Cardiol. 2019; 38. https://scielo.conicyt.cl/pdf/rchcardiol/v38n1/0718-8560-rchcardiol-38-01-00054.pdf.

40. Sociedad Argentina de Cardiología . Consenso de Cardio-Oncología. Rev. argent.cardiol. 2019; 87(5). https://www.sac.org.ar/wp-content/uploads/2019/12/consenso-87-5.pdf

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The authors declare that there is no conflict of interest.

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